Abstract Submitted
for the MAR12 Meeting of
The American Physical Society

Lysozyme Aggregation and Fibrillation Monitored by Dynamic Light Scattering LOUIS NEMZER, BRET FLANDERS, JEREMY SCHMIT, CHRISTOPHER SORENSEN, Department of Physics - Kansas State University — The aggregation of amyloidogenic proteins provides a rich phase space with significant biomedical implications, including a link with several age-related diseases. We employed dynamic light scattering to monitor the aggregation of lysozyme, a model protein, from a monomeric state until the formation of micron-sized fibrils. For an aqueous lysozyme solution buffered at pH 2, the auto-correlation function of the scattered light intensity was found to be well-fit by a single exponential function with decay time $\tau = 1/(2Dq^2) = 0.25$ ms, which corresponds to a mean hydrodynamic radius (R_H) of 2.2 nm, very likely generated by monomers. Ethanol (4% v/v final concentration) induced a partial unfolding, to $R_H = 4.6$ nm. The subsequent addition of 70 mM KCl was found to shrink the size back to $R_H = 2.5$ nm, as expected when a denatured protein refolds due to partial screening of the intramolecular repulsion. However, further aggregation was not observed. At pH 4, using a low-salt acetate buffer, more ethanol (10% v/v) was required to initiate unfolding, but once it occurred, larger aggregates formed. These results are consistent with the model that partial unfolding, which exposes beta-motif secondary structure, is a prerequisite for aggregation and fibrillation, but the aggregation fate depends on the protein charge state (pH) and screening (salt concentration).

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Date submitted: 11 Nov 2011 Electronic form version 1.4